CHAPTER 62

Examination of the Sensory System

KEY TEACHING POINTS

- For screening examinations, testing only the sensation of touch should suffice. In patients with sensory complaints involving large portions of the limbs or trunk, however, it is necessary to test all simple sensations (i.e., pain, temperature, vibration, touch) in order to uncover sensory dissociation (i.e., loss of one simple sensation but preservation of another)—an important clue to disease of the spinal cord.
- Diseases of the peripheral nerves, spinal cord, brainstem, and cerebral hemispheres each produce distinct sensory syndromes. These syndromes are distinguished by the distribution of sensory loss and the presence or absence of a sensory level, sensory dissociation, facial involvement, Horner syndrome, and weakness.
- The testing of cortical sensations (e.g., stereognosis, graphesthesia) requires preservation of the simple sensations. Abnormalities of cortical sensations indicate disease in the contralateral cerebral cortex.
- The lateral medullary stroke (Wallenberg syndrome) does not usually cause weakness but instead produces dramatic vertigo and loss of pain and temperature sensation on the ipsilateral face and contralateral body.

SIMPLE SENSATIONS

I. DEFINITIONS

There are four simple sensations: pain, temperature, touch, and vibration. These sensations are all called *simple* because their perception does not require a healthy contralateral cerebral cortex. Except for the sense of vibration, the simple sensations have distinct sensory organs in the skin; except for touch, their pathways in the spinal cord are well defined.

Hypesthesia refers to diminished ability to perceive a simple sensation; **anesthesia** refers to the complete inability to perceive a simple sensation. Although both hypesthesia and anesthesia originally referred only to the sensation of touch, many clinicians use these terms in reporting any of the simple sensations. **Hypalgesia** means that there is decreased sensitivity to painful stimuli; **analgesia**, complete insensitivity. **Hyperpathia**, **hyperesthesia**, and **allodynia** all refer to an increased sensitivity to sensory stimuli, often with unpleasant qualities, although some experts restrict hyperpathia to increased sensitivity from *painful* stimuli and allodynia to discomfort from *tactile* stimuli.

II. TECHNIQUE

The choice of sensory tests to be included in the physical examination depends on the clinical setting. For screening examinations of patients without sensory complaints, testing only touch sensation on all four extremities should suffice. If there are sensory complaints confined to one limb, testing for touch and pain sensation is usually performed, although testing pain sensation has a better chance of detecting subtle radiculopathies and peripheral nerve disorders.[1,2] (See the section on Dermatomes.) For screening diabetic feet and limbs at risk for neuropathic ulcers and arthropathy, clinicians should use Semmes-Weinstein monofilaments (see Chapter 55). Finally, for any patient with sensory complaints involving large portions of a limb or the trunk, testing for all simple sensations is necessary to uncover **sensory dissociation** (i.e., perception of one modality but not another)—a finding suggesting spinal cord disease. (See the section on Sensory Syndromes.)

During sensory testing, the patient's perceptions are compared with either a known standard of normal sensation (e.g., Semmes-Weinstein monofilaments for tactile sensation and tuning fork tests for vibratory sensation), the contralateral companion part of the patient's body, or the clinician's own sense of what is normal as gathered from previous experience.

A. TOUCH

The sensation of touch is usually tested qualitatively by stimulating the patient's skin lightly with a cotton swab, a piece of tissue paper, or the clinician's finger; it may also be tested quantitatively by using Semmes-Weinstein monofilaments (see Chapter 55).

B. PAIN AND TEMPERATURE

The usual techniques for testing pain sensation involve a safety pin bent at right angles or the sharp edge of a broken wooden applicator stick, both of which must be discarded after use to prevent the transmission of infection.[3] Because of the risk of transmitting infection, it is no longer permissible to use the built-in pin of many reflex hammers or the traditional tailor's pinwheel.

The traditional test for temperature sensation uses tubes of warm and cold water, although testing the patient's ability to distinguish the cold stem of a tuning fork from the warmer index finger is much simpler.[4]

C. VIBRATION

Vibratory sensation is tested with a tuning fork (usually 128 Hz; less often 256 Hz). There is no compelling reason for using one tuning fork over the other except that standards have been developed for the 128-Hz fork. Humans are most sensitive to vibration frequencies of 200 to 300 Hz and have difficulty consistently detecting frequencies below 100 Hz.[5,6] Traditionally the tuning fork is applied against a bony prominence, although this is based on the mistaken belief that bones contain the "vibration receptors"; vibratory sensation is just as good or even better over soft tissues without underlying bone (the clinician can easily demonstrate this by testing sensation on the abdominal wall).[7]

When a 128-Hz tuning fork is struck from a distance of 20 cm against the heel of the clinician's palm, a healthy 40-year-old person should perceive vibrations for at least 11 seconds when the stem of the fork is held against the lateral malleolus and for at least 15 seconds when it is held against the ulnar styloid.[8] These values decrease by 2 seconds for every decade of age greater than 40 years.

One disadvantage to vibratory testing is the fact that the vibrating impulse is conducted away from the tuning fork, thus preventing precise definition of sensory boundaries in patients with peripheral nerve injuries.[7]

Rumpf introduced the tuning fork to bedside neurology in 1889.[9]

III. CLINICAL SIGNIFICANCE

A. TOUCH, PAIN, AND TEMPERATURE SENSATION

Abnormalities of simple sensations define all important clinical sensory syndromes: peripheral nerve injury, radiculopathy, spinal cord syndromes, lateral medullary infarction, and thalamic and cerebral hemispheric syndromes. (See the section on Sensory Syndromes.) No diagnostic test has proved superior to bedside examination. The finding of diminished pain sensation (to safety-pin stimulus) detects the loss of small nerve fibers on skin biopsies with a sensitivity of 88%, specificity of 81%, positive LR = 4.6, and negative LR = 0.2;[10] the clinician's bedside assessment of hypesthesia is a more specific predictor of nerve fiber loss than that obtained with an automated touch-pressure esthesiometer.[11] Physical examination may even be superior to nerve conduction testing, a test of only the large myelinated peripheral nerve fibers, not the smaller unmyelinated fibers that carry pain and temperature sensations and from which many uncomfortable sensory syndromes originate.[12]

Diabetic feet insensate to the 5.07 monofilament are at increased risk for subsequent foot ulceration and amputation (see Chapter 55).

B. VIBRATORY SENSATION

Vibratory sensation is often diminished in peripheral neuropathy and spinal cord disease but spared in disease confined to the cerebral cortex.[7] Although it is a highly developed sensation—Helen Keller could interpret speech by feeling the vibrations of the speaker's larynx, lips, and nose—it lacks distinct sense organs and its neuroanatomic pathways remain obscure.[7,13] Traditionally it is associated with proprioception because impulses from both sensations ascend in the posterior columns of the spinal cord, but there are many clinical examples of dissociation of vibratory and proprioceptive loss, both in peripheral neuropathy and spinal cord disease.[7,14] (See the section on Proprioception.)

C. HYPERPATHIA AND ALLODYNIA ARE NONSPECIFIC FINDINGS

Hyperpathia and allodynia occur in many different painful conditions, including peripheral neuropathy, brainstem infarction, and thalamic stroke; by themselves they have no localizing value.[15,16]

PROPRIOCEPTION

I. DEFINITION

Proprioception allows individuals to detect joint motion and limb position when their eyes are closed.[17] Like most of the simple sensations, proprioception has distinct sense organs and ascending pathways in the spinal cord. Unlike simple

sensations, however, full perception requires a healthy contralateral cerebral cortex; in this way it resembles cortical sensations.[18,19] (See the section on Cortical Sensations.)

Sir Charles Bell originally called proprioception the "sixth sense." In 1906, Sherrington introduced the term "proprioception" to describe this sensation.[17,20]

II. TECHNIQUE

The conventional test of proprioception is to lightly hold the sides of the patient's finger or toe and bend it slowly up and down. The patient is asked to indicate any sensation of movement and the movement's direction. Because normal persons perceive motion much more easily than direction, a normal person may accurately indicate the presence of motion all the time but indicate the wrong direction up to 10% of the time.[21] Normal individuals can detect 1 to 2 degrees of movement in most joints, the hips being the most sensitive.[21,22]

Another test of proprioception examines the ability to direct a limb to a given point, again with eyes closed. In one version, the clinician positions the patient's outstretched index finger on his or her own index finger. The patient then drops the arm to the side and attempts to find the previous position. Normal individuals consistently come within 5 cm of the target.[20]

Patients with severe proprioceptive loss depend on vision for balance and thus become very unstable when they close their eyes or walk in darkness. This dependence on vision forms the basis for another test of proprioceptive loss, the Romberg sign, which is discussed fully in Chapter 7.

III. CLINICAL SIGNIFICANCE

Proprioceptive loss is common in peripheral neuropathy (e.g., diabetes mellitus), spinal cord disease (e.g., multiple sclerosis, vitamin B12 deficiency, tabes dorsalis), and severe hemispheric disease. In unilateral disease of the spinal cord (e.g., the Brown-Séquard syndrome), proprioception is lost on the side of weakness, opposite the side with pain and temperature loss. (See the section on Sensory Syndromes.) In patients with strokes, proprioceptive loss indicates extensive damage and correlates with a poorer functional recovery and higher mortality.[23]

According to traditional teachings, a disproportionate loss of vibration sensation and proprioception (compared with pain and temperature sensation) occurs in diseases of the dorsal columns of the spinal cord (e.g., tabes dorsalis, multiple sclerosis, vitamin B12 deficiency) and some peripheral neuropathies (e.g., diabetic polyneuropathy). Although this teaching is true, most patients with these disorders also have abnormalities of pain and temperature sensation.[7,24]

CORTICAL SENSATIONS

I. DEFINITION

Cortical sensations are those requiring higher integration and processing if they are to be perceived properly. Consequently perception of cortical sensations requires a healthy contralateral cerebral cortex. These sensations may become

abnormal in cerebral hemispheric disease *even though* the simple sensations are preserved.

II. TECHNIQUE

Testing for cortical sensations has three requirements: (1) the patient's eyes are closed, (2) the patient lacks dementia, and (3) most of the simple sensations, especially touch, are preserved. If the simple sensations are profoundly altered, as in severe peripheral neuropathy, no sensory information will reach the cerebral hemisphere and tests for cortical sensation become uninterpretable.

A. TWO-POINT DISCRIMINATION

Two-point discrimination is the ability to distinguish two compass points simultaneously applied to the skin. The normal minimal distance is 3 cm for the hand or foot and 0.6 cm for the fingertips.[14,18,25,26]

B. TACTILE RECOGNITION (STEREOGNOSIS)

Tactile recognition is the ability to recognize common objects such as a key, paper clip, coin, tweezers, or rubber ball placed in the hand. Normal individuals can name more than 90% of such objects within 5 seconds.[27,28]

C. GRAPHESTHESIA

Graphesthesia is the ability to identify letters or numbers traced on the hand or foot. Normal individuals can easily recognize symbols 1 cm in height on the fingertips and 6 cm high elsewhere.[18]

D. LOCALIZATION

Localization is the ability to accurately point to a spot on the body that has just been touched by the clinician.

E. BILATERAL SIMULTANEOUS TACTILE STIMULATION

This tests the patient's ability to recognize that both sides of the body are being touched simultaneously. The term **tactile extinction** refers to the patient's consistent failure to detect the stimulus on one side of the body.[29]

F. APPRECIATION OF WEIGHTS

Appreciation of weights is the ability to perceive differences in weight between two objects placed sequentially in the patient's hand. This test was used more often several decades ago than it is now.[30]

III. CLINICAL SIGNIFICANCE

Lesions of the posterior parietal lobe may preserve the simple sensations but eliminate proprioception and cortical sensations. The loss is typically confined to the contralateral distal parts of the limbs, sparing the face and trunk.[19,30-32]

It is important to note that cortical disease may also may eliminate any or all of the simple sensations, especially if the lesion involves the anterior parietal lobe (postcentral gyrus) or deeper white matter.[7,19,30,33] These lesions often cause a dense sensory loss on the opposite side of the body, involving the trunk, limbs, and face—a condition sometimes referred to as the **pseudothalamic syndrome** because of its resemblance to the sensory loss of thalamic disease. (See the section on Sensory Syndromes below.)[19]

DERMATOMES

I. DEFINITION

A **dermatome** defines the area of skin innervated by a single nerve root or spinal segment. Dermatomes are primarily used to determine whether the sensory loss on a limb corresponds to a single spinal segment, implying that the lesion affects that particular nerve root (i.e., radiculopathy), and to assign a neurologic "level" to a spinal cord lesion.

II. DERIVATION OF THE DERMATOMAL MAPS

The original human dermatomal maps emerged from Sherrington's experiments with monkeys and Head's observations of patients with herpes zoster infection.[2,34] These maps have since been revised on the basis of several types of evidence collected over the last century, including neurosurgical observations (by Cushing, Foester, and Keegan), experiments injecting novocaine next to the nerve roots of medical student volunteers, and electrical stimulation of the skin while recording potentials at the nerve roots.[1,2,34-37] Differences among dermatomal maps, which are minor and primarily deal with how far proximally some limb dermatomes extend, probably reflect biologic variation and differences in experimental method (i.e., sensory loss from a herniated disc or novocaine injection is not necessarily the same as that from root resection).

III. TECHNIQUE

The dermatomal map in Fig. 62.1 is the international standard used for classifying patients with spinal cord injury (Table 62.1).[38] Two principles apply in evaluating the dermatomal pattern of sensory loss. First, contiguous dermatomes overlap, which means that damage to one nerve root may cause either no anesthesia or a sensory loss confined to a small area. These small areas, which are referred to as **signature zones**, define the sensory level in patients with spinal cord disease.* Second, tactile dermatomes are larger than pain dermatomes. This suggests that when only one or two segments are affected, testing for pain sensibility is a more sensitive method of examination than testing for abnormal touch.[1,2]

IV. CLINICAL SIGNIFICANCE

A. THE SENSORY LEVEL IN SPINAL CORD DISEASE

The patient's sensory level is often several segments *below* the actual level of the lesion in the spine (e.g., the patient with a T8 sensory level may have a lesion in the T3 segment of the spinal cord).[39-42,†] There are two explanations for this

* In sensory testing as in motor testing, the "neurologic level" refers to the most caudal level with *normal* function rather than the first level with abnormal function. For example, a patient with sensation in the nipple line but none below it has a "T4 sensory level."

† In 1887, during the first successful operation to remove a spinal tumor, the surgeon's initial incision, which had been based on the patient's sensory level at T5, had to be revised upward twice before the tumor was found at the T2 level.[43]

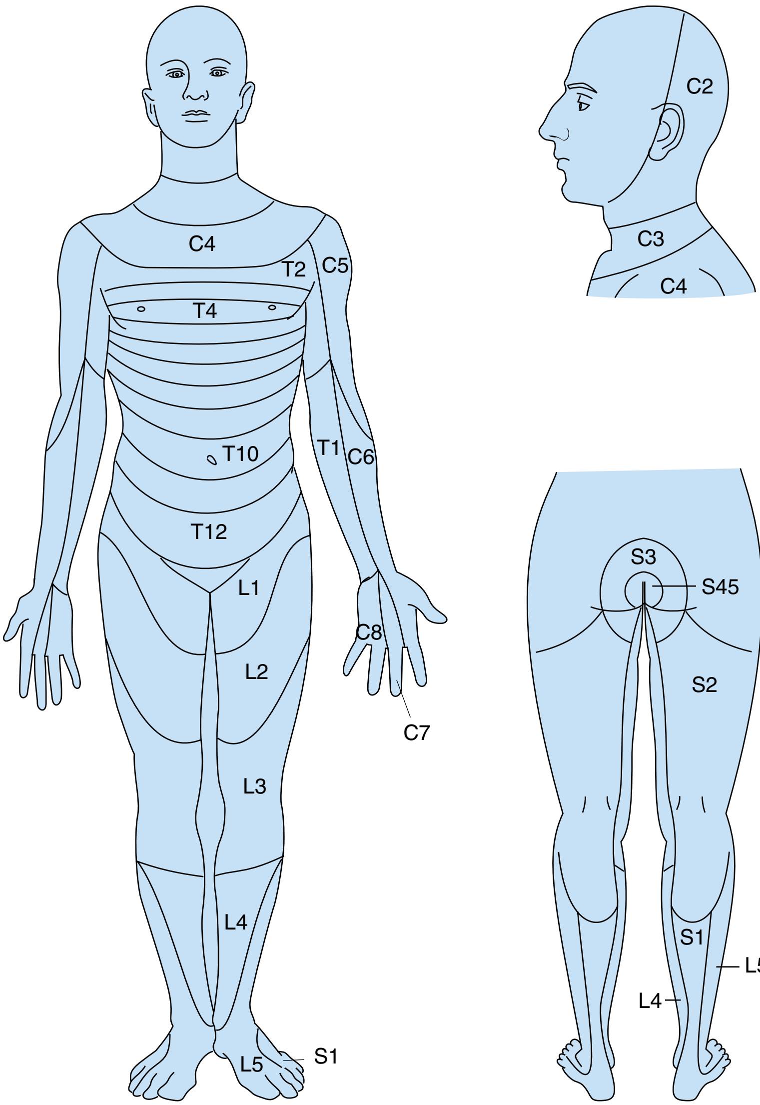

FIG. 62.1 DERMATOMES. This is the dermatome map recommended by the American Spinal Injury Association.[38] A printable copy is available at http://www.asia-spinalinjury.org/learning/. Note that the C2 dermatome includes the angle of the jaw and most of the ear. The precise boundaries of the S1 and S2 dermatomes are the most controversial.[35]

phenomenon: (1) The organization of the ascending spinothalamic pathway (carrying pain and temperature sensation) makes the more lateral fibers carrying lower body sensations more vulnerable to external injury. (2) Instead of directly damaging the contiguous cord, the spinal lesion causes injury at a more distant segment by compromising the cord's blood supply.[39,40]

TABLE 62.1 Dermatomes and Their Signature Zones

Spinal Level	Signature Zone
CERVICAL	
C3	Supraclavicular fossa
C4	Top of the acromioclavicular joint
C5	Lateral side of the antecubital fossa
C6	Thumb
C7	Middle finger
C8	Little finger
THORACIC (SELECTED LEVELS)	
T1	Medial (ulnar) side of the antecubital fossa
T2	Apex of axilla
T4	Fourth intercostal space (nipple line)
T10	Tenth intercostal space (umbilicus)
T12	Inguinal ligament at midpoint
LUMBAR	
L1	Half the distance between T12 and L2
L2	Midanterior thigh
L3	Medial femoral condyle
L4	Medial malleolus
L5	Dorsum of the foot at the third metatarsal phalangeal joint
SACRAL	
S1	Lateral heel
S2	Popliteal fossa in the midline
S3	Ischial tuberosity
S4-S5	Perianal level

Based on reference 38 and original work cited in text.

When the sensory and motor levels disagree, the motor level is a more reliable indicator of level of injury and future disability.[44] In some patients with spinal cord disease, the most accurate indicator of the spinal segment affected is the site of the patient's vertebral pain and tenderness or the level of the patient's radicular pain.[41,45]

B. DERMATOMAL LOSS IN RADICULOPATHY

The clinical significance of dermatomal sensory loss in disorders of the nerve roots is discussed in Chapter 64.

SENSORY SYNDROMES

I. TECHNIQUE

Fig. 62.2 depicts the sensory loss characteristic of the important sensory syndromes. Sensory loss confined to a *portion* of a limb suggests injury to a peripheral nerve, plexus, or spinal root, as discussed in Chapter 64. When sensory loss involves *most*

of a limb or the *trunk*, a systematic approach using the following questions defines the syndrome.

A. DOES THE SENSORY LOSS INVOLVE BOTH SIDES OF THE BODY?

Involvement of *both* sides indicates polyneuropathy or spinal cord disease. Involvement of *one* side indicates contralateral disease of the brainstem, thalamus, or cerebral cortex. In patients with pure hemisection of the spinal cord (i.e., Brown Séquard syndrome), there is sensory loss on both sides of the body, although pain and temperature sensation is lost on the side *opposite to* the lesion and tactile sensation is lost on the side *of* the lesion.

B. IS THERE A SENSORY LEVEL?

A sensory level is a distinct border on the trunk below which sensory testing is abnormal and above which it is normal. A sensory level indicates spinal cord disease, although the finding sometimes also occurs in lateral medullary infarction.[15,46-49]

C. IS THERE SENSORY DISSOCIATION?

Sensory dissociation is a disproportionate loss of one or more simple sensations with preservation of others. Loss of pain and temperature sensation with preservation of touch and vibratory sensation is a feature of some *incomplete* spinal cord syndromes (e.g., syringomyelia, spinal stroke, and Brown-Séquard syndrome).

D. IS THERE SENSORY LOSS ON THE FACE?

Sensory loss on the face indicates disease above the spinal cord—in the brainstem, thalamus, or cerebral hemispheres. In brainstem disease (e.g., lateral medullary syndrome), the sensory loss on the patient's face is *opposite* to the side of sensory loss on the body; in disease of the thalamus or cerebral hemisphere, the sensory losses on the face and body are on the *same* side.

E. ARE THERE ASSOCIATED NEUROLOGIC SIGNS?

Most disorders causing the sensory syndromes depicted in Fig. 62.2 also cause significant weakness (indicated by the *arrows* in Fig. 62.2), a major exception being the lateral medullary syndrome.

The presence of an associated **Horner syndrome** (see Chapter 21 for definition) indicates disease of the ipsilateral brainstem or cervical spinal cord.[50]

II. DEFINITION OF THE SENSORY SYNDROMES

Disorders of the peripheral nerves and spinal roots are discussed in Chapter 64.

A. POLYNEUROPATHY

Polyneuropathy is a bilateral **stocking-glove** sensory loss that spares the face (the sensory loss resembles the pattern of a stocking or glove because polyneuropathy affects all nerves of the same length equally). Because the sensory loss of polyneuropathy affects the longest nerves first, hypesthesia initially appears in the feet, later in the fingertips, and—only after extensive involvement of the arms and legs—finally in the anterior trunk.[51] Atrophy of the small muscles of the feet and hands and absent ankle reflexes are common. Distal weakness may occur, but because the nerves to the foot dorsiflexors are longer than those to plantarflexors, patients with polyneuropathy have more trouble walking on their heels than on their toes (the opposite finding, trouble walking on the toes but not on the heels, suggests an alternative diagnosis).[52]

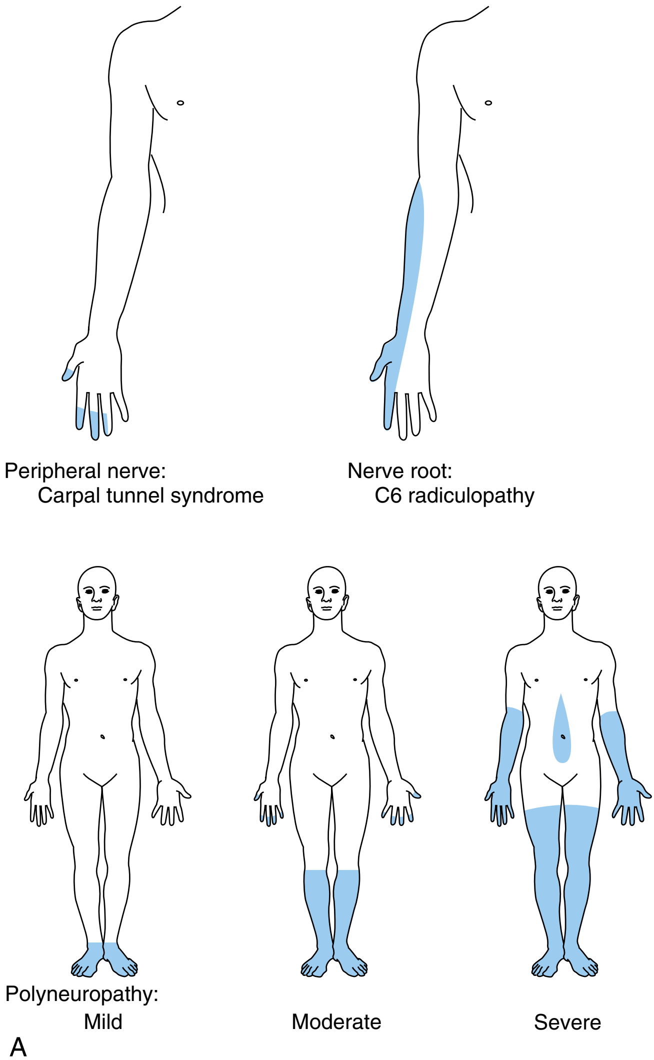

FIG. 62.2 SENSORY SYNDROMES. In these figures (A and B), the blue shading indicates hypalgesia (loss of pain sensation) and the *arrows* indicate limbs with significant accompanying weakness. In the Brown-Séquard syndrome (hemisection of the cord, *top row*, Fig. 62.2B), there is often diminished tactile sensation on the side of weakness and opposite the side with hypalgesia.

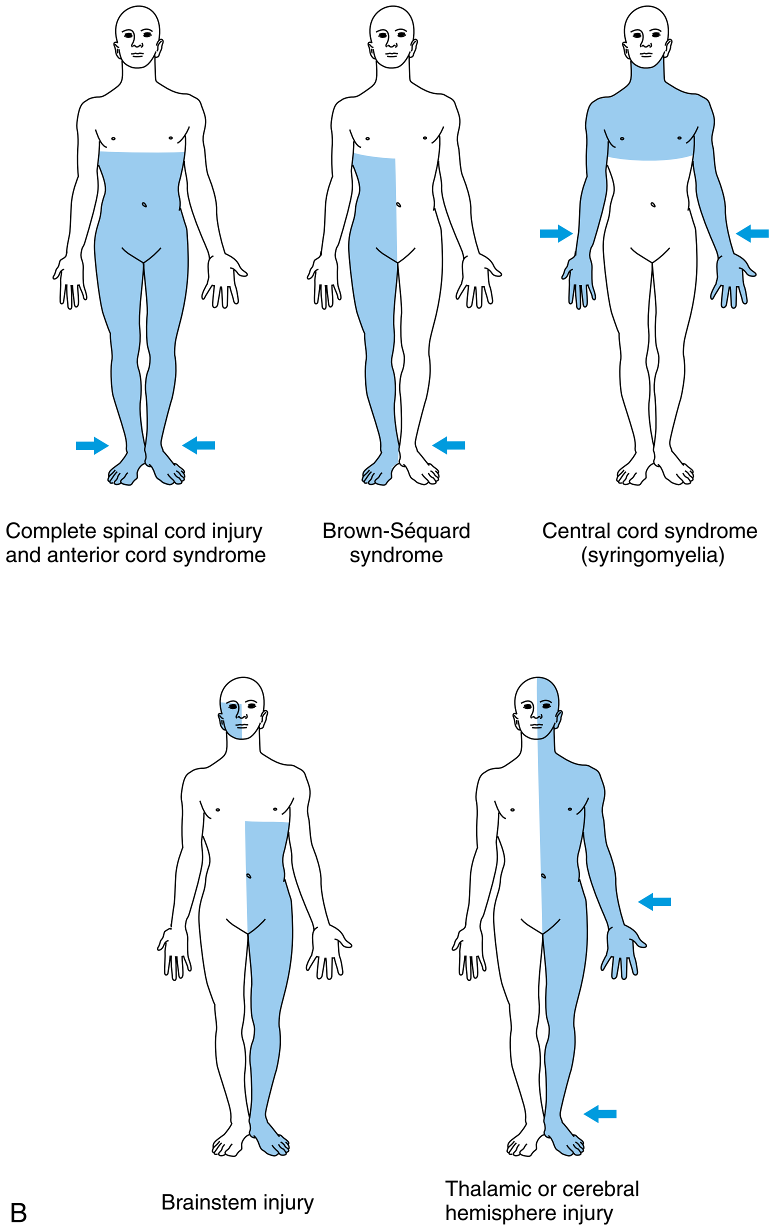

FIG 62.2, CONT'D

B. SPINAL CORD SYNDROMES

1. COMPLETE SPINAL CORD LESION

A **complete spinal cord lesion** causes a sensory level with loss of all simple sensations below that level, weakness (tetraparesis or paraparesis), and urinary retention.

2. INCOMPLETE SPINAL CORD LESIONS

A. ANTERIOR CORD SYNDROME

Spinal stroke, which may follow prolonged hypotension or trauma to the aorta, resembles the complete spinal cord lesion except that there is a disproportionate loss of pain and temperature sensation and relative sparing of touch and vibration owing to the more vulnerable blood supply of the ventral cord.[53]

B. BROWN-SÉQUARD SYNDROME

Brown-Séquard syndrome describes injury to one-half of the cord, causing *contralateral* loss of pain and temperature sensation but *ipsilateral* paralysis and diminished touch sensation.[50] Unilateral disease of the cervicothoracic region may involve the ascending sympathetic fibers and cause an ipsilateral Horner syndrome.[50]

The pure Brown-Séquard syndrome is rare. Instead, most patients with unilateral disease of the spinal cord present with bilateral weakness and sensory loss, although the weakness is greatest on the side of the lesion and the hypalgesia is greatest opposite the lesion.[50]

C. CENTRAL CORD SYNDROME

In syringomyelia, the sensory loss typically involves one or both arms. Some 75% of patients have atrophy and weakness of one or both hands or sternocleidomastoid muscles.[54,55]

C. LATERAL MEDULLARY INFARCTION

Wallenberg syndrome is a dramatic syndrome presenting with dizziness and sensory loss on opposite sides of face and body but no weakness (the lesion is ipsilateral to the facial analgesia). Common associated signs are diminished corneal reflex, ipsilateral limb ataxia, nystagmus, ipsilateral Horner syndrome, gait ataxia, and ipsilateral palate weakness (Table 62.2).

D. THALAMIC DISEASE

A lesion in the thalamus may cause loss of all simple sensations on the opposite side of the body in association with hemiparesis, vertical gaze abnormalities, miosis, and aphasia.[33,63,64]

E. CEREBRAL HEMISPHERIC DISEASE

Cerebral hemispheric disease may cause a dense sensory loss and hemiparesis identical to that of thalamic disease (**pseudothalamic syndrome**)[19] or the selective loss of cortical sensations in the distal parts of the extremities. (See the section on Cortical Sensations.)

TABLE 62.2 Lateral Medullary Infarction (Wallenberg Syndrome)

Physical Finding	Frequency (%)*
CRANIAL NERVES	
Diminished corneal reflex (V and VII)	91
Ipsilateral Horner syndrome†	41-95
Ipsilateral face analgesia (V)	50-86
Nystagmus	56-100
Ipsilateral palate weakness (IX, X)	52-86
Ipsilateral facial weakness (VII)	18-43
SENSORY	
Contralateral body analgesia	88
COORDINATION	
Ipsilateral limb ataxia	55-95
Gait ataxia	91

*Results are overall mean frequency or, if statistically heterogeneous, the range of values.
†Strictly speaking, Horner syndrome does not involve cranial nerves, although it is discovered during examination of the pupils and eyelids.
Data obtained from 485 patients based upon references 48, 56-62.

The references for this chapter can be found on www.expertconsult.com.

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